THE RHODE ISLAND MEDICAL JOURNAL

The Official Organ of the Rhode Island Medical Society
Issued Monthly under the direction of the Publication Committee

VOLUME XIX
NUMBER 11 | Whole No. 326 | PROVIDENCE, R. I., NOVEMBER, 1936 | PER YEAR $2.00 SINGLE COPY 25 CENTS

THE SCHILLING HEMOGRAM IN APPENDICITIS*

HENRI E. GAUTHIER, M.D.
34 HAMLET AVENUE, WOONSOCKET, R. I.

The conception of this unique arrangement of the polymorphonuclear neutrophiles, of which Schilling was the originator, dates back to the year 1909. This idea was substantiated by several papers explaining the new arrangement and in 1912, the first edition of his book, devoted to tropical diseases, was published. Later, the Hemogram was applied to General Medicine and has proved to be of great value because of its symptomatic, diagnostic and even prognostic significance. In 1929, the Seventh and Eighth editions were translated into English by Gradwohl of St. Louis and within two years, this English translation was completely exhausted.

The text-books generally give scant and indefinite information concerning the clinical laboratory findings in Appendicitis. The authors fairly well agree, however, on the importance of a leucocyte count, though some consider it of comparatively little value, others consider it of equal value to the pulse and temperature. We are informed further that the leucocyte counts may vary considerably, that a count of 30,000 leucocytes may indicate a gangrenous appendicitis, whereas, in a patient with a lowered resistance and with an inflammatory process just as severe, the leucocyte count may be only 2,000. This is remindful of the saying that "A competent theologian never sins because he is able to find loopholes to every conceivable accusation of sin." Mention is further made that there is a relative increase of the polymorphonuclear neutrophiles and even such increase is of very little help at times in making a diagnosis. For the surgeon who relies on the history, symptoms and the aspect of the abdomen for his diagnosis, a leucocyte count and an increase of the polymorphonuclear neutrophiles will constitute conclusive proof that an operation is indicated. Should neither of these fit into the clinical picture, it might be to his advantage to disregard them altogether.

There is no infallibility in the diagnosis of appendicitis. All the information possible must be weighed carefully. The diagnosis is very simple when the history, symptomology, objective signs and laboratory findings fit the text-book picture, but when confronted with a case which is at variance with this slide rule, the diagnosis then becomes either one of chance, or one of analytical dissection of its every phase. The diagnosis of appendicitis must be more than a simple diagnosis; it must be inclusive to the point of being informative on the degree, severity and the extension of the inflammation present and in view of a more critical clientele, it must in itself prognosticate on the chances, immediate and remote, for recovery. Obviously, an estimation of the true underlying condition is far more difficult and demands meticulous observation of all the evidence gathered.

It is my intention to view the Schilling Hemogram from a surgeon's standpoint and not as would a skilled pathologist or hematologist. It would be presumptuous to expect a masterful interpretation of the Schilling Hemogram on the part of a surgeon and therefore only those hap-hazard observations made in the course of ordinary surgical practice and deemed worthy of mention, will be dwelt upon.

To understand fully the definition of the Schilling Hemogram, one must be familiar with the Ehrlich Differential Blood Count. Up to a few years ago, when a differential white blood count was requested, the Ehrlich differential count was made and the cells were grouped as follows:

Basophiles	0.5%
Eosinophiles	2-3%
Polymorphonuclear Neutrophiles	60-65%
Large Lymphocytes	5-10%
Small Lymphocytes	20-25%
Monocytes	3-5%

In the Schilling Hemogram, the basophiles, the eosinophiles, the lymphocytes and the monocytes have been retained, but the polymorphonuclear neutrophiles have been further divided into the segmented and non-segmented cells, namely, the

*Read before the One-hundred and twenty-fifth Annual Meeting of the Rhode Island Medical Society, Providence, June 3 and 4, 1936.

segmented nuclears as the mature polymorphonuclear neutrophiles and the non-segmented cells grouped according to the following nomenclature —the stab nuclears or band forms as mature non-segmented cells; the myelocytes and juveniles as the immature non-segmented cells.

The presence of the myelocyte indicates hyperactivity or marked regeneration. In large numbers, it confirms the diagnosis of myelogenous leukemia. It is never to be found except in very grave conditions. The juvenile cell is an immature polymorphonuclear neutrophile, is rarely in the peripheral blood system and its presence is significant of regeneration. The stab nuclear or band cell is a mature cell, lacking segments. An increase means degeneration, grave infection or poor resistance. The segmented nuclear is the mature segmented polymorphonuclear neutrophile of the original Ehrlich differential group. All these cells have their origin in the bone-marrow.

An increase in the lymphocytes is indicative of either rejuvenation or irritation. After a long and severe illness, a steady increase is proof of healing.

The significance of the monocytes is not altogether clear. They increase with infection and are held and believed to be the last cells to return to normal following infection.

An increase of the non-segmented stab or band forms above the normal limit and the appearance of the juvenile cells constitute a shift to the left, the greater the shift the more severe the infection. In some cases this shift is rapid and pronounced while in others, even though the infection is severe, the shift may be moderate and may be entirely absent. As in the Ehrlich differential, infection results in an early increase in the polymorphonuclear neutrophiles. Should the infection be trifling, these cells are moderately increased, whereas in the presence of severe infection, greater reaction follows and the bone marrow, if capable, will send out all the available polymorphonuclear neutrophiles and should these not suffice, the immature cells must respond. Therefore, it is evident that the segmented forms act as a lever, an increase on the left, occurring in acute infections, interpreted as a shift to the left, while an increase on the right, occurring in chronic infections and during convalescence of severe acute diseases, is interpreted as a shift to the right for the former and as a healing phase for the latter.

The degree of shift to the left is expressed by the Nuclear Index, which is normally over ten. It is obtained by dividing the number of non-segmented forms into the number of segmented forms. This index may be normal or above normal even in the presence of infection, when the non-segmented forms have failed to increase because of lack of reaction, lack of resistance or because of sufficient segmented forms. When the non-segmented forms are as great or greater in number than the segmented forms, the nuclear index is 1or —0 and such a condition is usually of grave prognosis and very often fatal. It is referred to as an Agonal Picture.

The basophiles are normal up to 1%. The eosinophiles are present from 2 to 4%. In the normal Hemogram, the myelocytes are absent. Though normally absent, the juvenile neutrophiles may be present up to 1%. The normal range for the stab nuclear is from 3 to 5%, while the segmented nuclears are present in normal limits from 51 to 67%. The lymphocytes are normal in number if they are within 21 to 35% and the monocytes average from 4 to 8%. In the normal Hemogram there is no shift, the Nuclear Index being over 10.

At first sight, some of the claims of the Schilling Hemogram sounded very mythical, but they drew more interest when the cell arrangement was found to fit the operative findings and the pathological reports in a surprising percentage of cases. As previously stated, the Schilling Hemogram has not been found to be infallible, but through it more information has been derived from laboratory findings than heretofore. There has been less hesitancy to operate upon the nervous patient who does much to conceal his symptoms and a better understanding of the muscular abdomen is possible. Every surgeon has had at some time a nervous patient who refused to admit nausea, vomiting, tenderness or pain, with the result that a diagnosis of appendicitis was dispelled, only to be greatly surprised on the subsequent visit by the presence of a mass in the right lower abdominal quadrant. On the first visit, the disease was probably in an early stage; had a blood count been insisted upon, it would undoubtedly have helped to give the desired information. Schilling appropriately says that: "Clinical observation of a nervous and somewhat excitable patient often inclines us to be led astray, in spite of our most careful investigations." For the past six years, every suspected case of appendicitis in my

practice was referred for a complete blood count and a Schilling differential blood count. With each case, my interest in the Schilling Hemogram became more intense. Having gained some knowledge of the fundamental principles of the Schilling Hemogram, I have collected 148 cases which have been classified and will be presented in groups. Individually, a discrepancy occasionally appears, which may be caused by any of several factors.

Table I

CHRONIC-CYSTIC, DEGENERATIVE OR OBLITERATIVE
NEUTROPHILES

Case	W.B.C.	Eos.	Ju.	Stabs.	Seg.	Lym.	Mon.	% Neut.	N.I.
1	14,000	.....	5	2	79	14	.....	86	11.2
2	18,000	.....	6	5	63	21	5	74	5.8
3	8,500	.....	3	3	57	32	5	63	9.5
4	9,200	.....	3	5	67	24	1	75	8.3
5	10,000	1	10	3	40	46	....	53	2.8
6	5,200	3	1	2	67	25	2	70	22.3
7	14,000	1	3	2	76	15	3	81	15.2
8	12,600	.....	5	2	83	5	5	90	11.8
9	10,500	.....	3	2	65	24	6	70	13
10	6,500	2	.....	4	70	24	....	74	17.5
11	6,500	.....	5	4	65	24	2	74	7.2
12	9,050	1	4	5	66	21	3	75	7.3
13	13,000	.....	4	.....	78	14	4	82	19.5
14	18,000	.....	3	6	79	8	4	88	8.7
15	10,000	.....	7	1	76	15	1	84	9.5
16	18,500	3	10	3	50	30	4	63	3.8
17	20,000	.....	5	11	77	6	1	93	4.8
18	9,500	1	.....	4	81	9	5	85	20.2
19	7,500	.....	6	.....	73	20	1	79	12.1
20	14,000	2	6	3	74	11	4	83	6.6
Avge.	11,727	0.7	4.4	3.3	69.4	19.4	2.8	77	10.5
Range	5,200 20,000	0-3	0-10	0-11	40-83	5-46	0-6	53-93	2.8-22.3

The first group consists of so-called chronic cases, namely—Simple Chronic, Chronic Cystic, Chronic Degenerative and Chronic Obliterative Appendices. In these groups the microscopical pathological diagnosis has been used as a basis for the classification. A wide range is found in the leucocyte count — from 5,200 to 20,000, with an average leucocyte count of 11,727. The eosinophiles have kept within normal limits. With the exception of two cases, numbers 5 and 16, the juveniles have displayed only a moderate increase with an average of 4.4 for the group. The stab nuclears are within the normal limits, except case number 14, which has a count of 6, and case number 17, a count of 11. The average obtained for the stab nuclear cells is normal. The segments show a decrease below normal in case number 5, which has a count of 40, otherwise, they show varying degrees of increase to 83%, for an average of 69.4. The lymphocytes range from 5 to 46 and average 19.4. Only in case number 5 is the count higher than normal. The monocytes remain within a normal range. The percentage neutrophiles averages 77% and ranges from 53 to 93%. The nuclear index varies greatly from 2.8 to 22.3. A low nuclear index with considerable shift to the left is present in cases 17, 16, 2, and 5. In estimating the need for operative intervention, one must evaluate in order the number of white blood cells, juveniles, stabs, segments, the percentage neutrophiles and the nuclear index.

Table II

SUB-ACUTE APPENDICES
NEUTROPHILES

Case	W.B.C.	Eos.	Ju.	Stabs.	Seg.	Lym.	Mon.	% Neut.	N.I.
1	5,200	.....	6	1	61	26	6	68	8.7
2	22,800	.....	.....	16	59	12	13	75	3.7
3	11,400	3	....	10	71	15	1	81	7.1
4	18,000	.....	2	1	81	7	9	84	27
5	7,500	.....	....	4	87	7	2	91	21.7
6	14,500	.....	3	2	59	24	12	64	11.8
7	10,500	.....	4	6	70	15	5	80	7
8	20,000	.....	4	2	91	3	....	97	16.1
9	13,500	.....	4	.....	64	26	6	68	16
10	5,400	1	.....	6	59	25	9	65	9.8
11	14,600	.....	....	14	72	7	7	86	5.1
12	16,000	.....	5	6	82	3	4	93	7.4
13	10.200	2	.....	8	66	20	4	74	8.2
14	20,000	.....	....	2	75	15	8	77	37.5
15	16,000	4	7	10	59	12	8	76	3.4
16	8,500	.....	7	5	64	20	4	76	5.3
17	14,200	.....	8	.....	80	9	3	88	10
18	9,300	.....	2	3	85	10	....	90	17
19	8,500	.....	.....	2	76	12	10	78	38
20	9,500	.....	.....	4	74	16	6	78	18.5
21	16,000	2	.....	3	80	12	3	83	26.6
22	16,400	2	6	5	70	12	5	81	6.3
23	23,000	1	2	4	78	12	3	84	13
24	8,400	1	1	3	78	16	1	82	18.7
25	12,900	1	3	2	92	2	....	97	18.4
26	6,500	.....	....	5	63	31	1	68	12.6
27	9,500	.....	2	4	72	18	4	78	12
28	12,000	.....	5	3	71	15	6	79	8.8
29	11,000	.....	2	7	72	15	4	81	8
30	10,500	....	5	8	73	14	....	86	5.6
31	9,000	2	2	5	54	30	7	61	7.7
32	8,400	....	5	2	60	29	4	67	8.5
Avge.	12,475	.59	2.6	4.8	71.8	15.3	4.9	79.25	13.31
Range	5,200 23,000	0-14	0-8	0-16	54-92	2-31	0-13	61-97	3.4-38

A total of thirty-two cases of sub-acute appendicitis have been collected to form table 2. It will be seen that the leucocyte count and the component cell groups have a greater range than was present in the chronic appendices. The leucocyte average of 12,475, also is greater. The eosinophiles remain within a normal zone. The juveniles, in most cases are either absent or very moderately increased, the greatest count being 8%. In none of these cases is there a pronounced stab shift. In fact, of the thirty-two cases, in only ten is an increase above normal seen, the highest count being 16% found in case number 2. A wide range is met with in the segment column, being from 54 to 92 with an average of 71.8. The lymphocytes range considerably, depending upon the degree of shift, in no case, however, are they found to be above normal. The average lymphocyte count is 15.3. In the monocyte column, there are 5 cases with an increase above normal, the highest count is 13 and the average 4.9. The range in percentage of the neutrophiles extends as high as 97% with a low of 61%, the average being slightly higher than in the preceding table. The nuclear index, because of the higher average obtained, is indicative of less shifting to the left.

Table III

ACUTE APPENDICES
NEUTROPHILES

Case	W.B.C.	Eos.	Ju.	Stabs.	Seg.	Lym.	Mon.	% Neut.	N.I.
1	17,500	1	.....	8	84	5	2	92	10.5
2	20,500	.....	4	2	82	12	.....	88	13.6
3	14,000	.....	4	16	75	3	2	95	3.7
4	30,000	.....	.....	4	92	1	3	96	25.5
5	16,000	.....	7	29	56	8	.....	92	1.5
6	13,900	4	5	8	71	12	.....	84	5.4
7	26,000	.....	1	10	87	2	.....	98	7.9
8	14,500	.....	2	5	84	9	.....	91	12
9	13,500	1	3	3	81	12	.....	87	13.5
10	32,000	.....	2	9	78	9	2	89	7
11	14,000	.....	1	17	70	12	.....	88	3.8
12	12,200	.....	2	6	80	12	.....	88	10
13	22,400	6	6	6	63	16	3	75	5.2
14	17,000	.....	.....	9	85	6	.....	94	9.3
15	8,950	.....	2	10	78	10	.....	90	6.5
16	26,600	.....	6	4	74	12	4	84	7.4
17	28,200	.....	.....	16	77	1	6	93	4.9
18	10,000	.....	3	16	72	6	3	91	3.2
19	18,500	.....	6	7	84	2	1	97	6.4
20	10,600	.....	.....	40	58	2	.....	98	1.4
21	15,450	.....	2	17	68	11	2	87	3.1
22	20,000	.....	.....	9	84	7	.....	93	9.3
23	23,000	.....	2	6	85	2	5	93	10.6
24	20,500	.....	3	7	83	4	3	93	8.3
25	16,500	.....	5	12	63	14	6	80	3.7
26	18,600	.....	3	17	67	5	8	87	3.3
27	10,600	.....	4	6	74	11	5	84	7.4
28	9,800	.....	10	10	62	14	4	82	4.1
29	16,800	2	3	6	73	12	4	82	8.1
30	16,000	.....	6	14	75	2	3	95	3.7
31	9,000	1	2	9	72	14	2	83	6.5
32	22,800	.....	2	12	83	3	.....	97	5.9
33	20,000	.....	9	5	79	7	.....	93	5.6
34	16,000	2	4	5	72	14	3	81	8
35	12,000	3	.....	30	49	12	6	79	1.6
36	18,000	.....	5	10	73	12	.....	88	4.8
37	22,000	.....	2	8	66	24	.....	76	6.6
Avge.	17,659	.54	3.1	11.02	74.5	8.6	2	88.7	7.0
Range	8,950 32,000	0-6	0-10	2-40	49-92	1-24	0-9	75-98	1.4-25.5

Table 3 represents the findings in thirty-seven acute appendices. It shows a substantial increase throughout in comparison with the findings of the two previous diagrams. The average of the white blood cells being 17,659 is 5,000 cells more than the average obtained in the sub-acute appendices. There is nothing abnormal in the eosinophile column except in case number 13, where a count of six is present. A perceptible increase is found in the juveniles, both in the average and the range. The average stab count is almost tripled and the highest count is 40, this occurring in case number 20 where the white blood cell count is only 10,600 with a neutrophilic count of 98%. Except for the two lymphocytes it would have been a total neutrophilic count. The shift in this case is decidedly degenerative. The average segment count is slightly higher than in the last diagram while, in its range, the low only goes down to 49 compared to 44 in the sub-acute appendices and the high is the same, 92. There is a decrease, both in the lymphocytes and the monocytes which is suggestive of a greater shift to the left. The percentage of neutrophiles is increased in its average by ten points and the range is maintained much higher. The nuclear index is decreased by half over that of the preceding table, which is evidence again of a greater shift.

Table IV

ACUTE GANGRENOUS APPENDICES—NON-PERFORATED
NEUTROPHILES

Case No.	Age	Sex	White Blood Cells	Juve-niles	Stabs.	Seg-ments	Lym-pho-cytes	Mono-cytes	% Neu-tro-philes	Nu-clear Index
1	2½	M	16,000	7	16	54	19	4	77	2
2	11	M	18,000	2	10	84	1	3	96	7
3	15	M	14,000	3	19	70	8	0	92	3
4	22	F	14,500	11	11	63	13	2	85	2
4	6 hrs. later		16,400	7	8	72	9	4	87	5
5	25	M	16,000	.....	2	86	6	6	88	43
5	Next day		17,000	2	6	86	6	.....	94	10
6	26	M	10,400	4	12	69	11	4	85	3.7
6	Next day		16,000	12	5	72	8	3	89	4
7	42	M	18,000	.....	14	76	9	1	90	5
7	6 hrs. later		18,950	5	10	78	7	.....	93	5
8	43	F	24,000	6	11	79	4	.....	96	4
Average			16,604	4.9	10.3	74.08	8.4	2.2	90.5	3.7
Range			10,400 24,000	0-12	2-19	54-86	1-19	0-6	77-96	2-7

There are eight non-perforated acute gangrenous appendices worthy of mention. It may be well to state at this time that blood counts should be made early and repeated at four to six hour intervals in doubtful cases. In this group, the white blood cells have a lower average and the range is not as great. There is an increase in the number of juveniles both in the average and the range. The stabs average slightly less than in the foregoing diagram and the range does not reach such a high level. The segments, lymphocytes and monocytes maintain about the same average as was found in the cases analyzed under acute appendices and their ranges are not as pronounced. The percentage of neutrophiles attains a higher average but the range is confined to a shorter extension. The nuclear index is half that of the acute appendices and the range is from 2 to 7.

(*Continued in the December Number*)

Fellows of the Rhode Island Medical Society who have not received the special invitation to attend the meeting of the Southern Medical Association, to be held at Baltimore on November 17-20, should consider the general invitation which has been printed in several issues of the JOURNAL, in the advertising section. "Regardless of what any physician may be interested in, regardless of how general or how limited his interest, there will be at Baltimore a program to challenge that interest and make it worth while for him to attend." A complete program may be obtained by addressing the Southern Medical Association, Empire Building, Birmingham, Alabama.

CORONARY THROMBOSIS*

ROGER I. LEE, M.D.
264 BEACON STREET, BOSTON, MASS.

In a very amazing fashion, coronary thrombosis has within a few years become familiar in medical speech. It happened to be one of those medical entities which, when it was delivered to us by Dr. J. B. Herrick, was readily and completely accepted as a matter of course. One is reminded of the earlier situation in regard to duodenal ulcer. Thirty years ago duodenal ulcer was supposed to be rather rare. In the case of duodenal ulcer, we had an instrument of precision, the X-ray, through the rapid development of which the easy diagnosis of duodenal ulcer became possible. In the case of coronary thrombosis, the electrocardiograph has facilitated the diagnosis in many obscure instances but has never played the essential role in diagnosis that the X-ray does in duodenal ulcer.

If one attempts to develop further the outstanding pathology of coronary thrombosis and to elucidate the etiologic factors which underlie the thrombosis, he comes to an impasse. We recognize clinically and pathologically the essential features of this condition and yet approach no closer than vague suspicion to the underlying causation. It is fashionable to say that any vascular disease represents the toll that the so-called stress and strain of modern life puts upon individuals. Such generalities strike me as inane and in many instances inaccurate. It is difficult to discover any factual data which are in the slightest convincing but it is extremely easy to get sentimental about vascular disturbances. If one brings forth the incidence of coronary thrombosis in individuals who have lived at what might be fairly called only first or second speed, the usual rejoinder is that it is the internal nervous sensitiveness of the individual which made his hillocks look like mountains. This reasoning seems to me unprofitable. One of the facts which does stand out is that there is a somewhat rough and general association in families of a tendency to vascular disease. A second fact is that this condition is much more common in men than it is in women. The third fact is that frequently but not always coronary thrombosis is associated with other vascular changes such as hypertension.

*Read at Providence, R. I., April 6, 1936, before the Providence Medical Association.

On the other side of the picture, we find the impression that the disease is on the increase and that the age incidence is apparently being lowered. We see cases of coronary thrombosis as early as the twenties and very rarely in the teens, occasionally in the thirties and frequently in the late forties. Because we have assumed that coronary thrombosis denotes old age it has startled us to realize that the condition occurs not infrequently in relatively early adult life.

We are in a haze in our views about the relation of cholesterol to this form of arteriosclerosis. We know that this is a rather highly selective form of arterial damage and that peripheral pipe stem arteries have no significance insofar as the coronary arteries are concerned. At one time, it was quite fashionable to take X-ray pictures of the legs of individuals with the notion of discovering the incidence of calcification in the arteries and possibly some index of the general arteriosclerosis. There was an attempt to link this to certain dietetic abnormalities such as the high fat diet which was so frequently obtained in diabetics before insulin was discovered. The fact that people with diabetes and on a high fat diet have more calcification in the arteries of their legs proves only that they have calcification in the arteries of their legs. It does not prove that calcification in the arteries of their legs is harmful. It does not prove that because they have calcification in the arteries of their legs they have arteriosclerosis anywhere else in the body. It certainly does not prove that the high fat diet necessarily had anything to do with it.

Somewhat over fifteen years ago, some X-rays were taken on both knees of a patient of mine. He was then in the sunny fifties and had wrenched his knee playing tennis. The X-ray showed nothing wrong with the structure of the knee joint but an extensive calcification of all of the blood vessels of his legs. The X-ray man thought that he should have a great deal of pain and discomfort in his legs but he had none at that time except the disability incident to the strained knee. He is now crowding seventy and is still playing tennis.

The uncle of this patient, a gentleman well over seventy, fell and fractured his popliteal artery. This required the intervention of a surgeon who found the popliteal artery entirely calcified and was at a good deal of bother to find a satisfactory place to tie the artery. Despite the gloomiest of prognostications, the old gentleman got along for a couple

of years without an amputation of his leg despite the fact that his popliteal artery was tied. One cannot help wondering if the localization of some types of arteriosclerosis is not under family influences. This is only an isolated observation and I put no stress except on its negative value. The occurrence of calcification in the vessels in the leg does not indicate the presence of any disturbance of the coronary artery that leads to coronary thrombosis.

Coronary thrombosis is part and parcel of angina pectoris. Angina pectoris is the major symptom of coronary thrombosis but not every patient with angina pectoris will have coronary thrombosis. I was called some thirty miles out of town to see a man who had been into Boston the day before for his annual or semi-annual overhauling. The doctor who had seen him was one of our most competent internists. The patient was in the early sixties and ever since his youth had had attacks, chiefly but not exclusively on exertion, which he called angina. They were rarely severe and were accompanied by no objective findings. Although the patient was supposed to be nervous and emotional, he had filled very satisfactorily an important position as teacher in a boys' school. The pain had rarely if ever interfered with his teaching. From his description it was substernal pain of varying intensity. There were periods when it was nearly constant and rather severe and other periods when it was inconsequential and there were in these periods waves of greater and lesser intensity. When I arrived, the patient was obviously excited. He looked the part of a well fed, rather robust man in good health. He had a little fever, a tachycardia and the sounds of his heart were poor. I felt that he had a coronary thrombosis, despite the years of angina of a very questionable variety. His brother-in-law, a doctor, sighed somewhat heavily when I gave my opinion and remarked "Of course, we have been through this many times before." This patient died a little over twenty-fours hours later, the death of coronary thrombosis.

Another case was that of a man well over seventy when he died, who had had for over forty years something that he called angina. The doctor who had taken care of him for forty years before I saw him was living and I had seen him for a half a dozen years or so. The doctor said, "I gave up the practice of medicine when I was eighty and I cannot now tell the difference between true and false angina any better than I could when I started the practice of medicine. If there is any such thing as false angina, this man has it and will die of something else." Curiously enough, however, his angina got rather more definite and precise. He finally died in an attack which seemed like nothing except coronary thrombosis. There was no autopsy and in neither of these cases were there electrocardiograms. I state these cases at considerable length as suggestions that coronary thrombosis occasionally does not come out of a clear sky but happens in patients who have had what has been known as false or nervous angina. The early, very mild, seemingly trifling attacks were precursors of something which was very much more serious.

There is no case which is more satisfactory when it comes up for discussion at the autopsy table than that of coronary thrombosis. Autopsy discussions always seem to me to be helpful but often unfair because there is an undisputable fact in the solution of the diagnostic mystery, namely, that the patient is dead. How different it is in clinical medicine. We see a living man. He is of the right age, he is apprehensive and has pain. The pains may not be entirely typical but they will do. What are we going to do about it? What are we going to call it? The situation is complicated by the fact that not only is the patient apprehensive but his family is much more so. There is nothing that one can do to establish the diagnosis with certainty at that time. Although I know that the patient has coronary thrombosis and consequently have an electrocardiogram taken, my experience is that the finding at that time is usually normal. After he has gone through the long complicated illness and is able to come to the office, the electrocardiogram then shows the characteristic changes. I know of no rule of thumb by which a diagnosis can surely be made. I have seen gall stones, gastric ulcer, gastric cancer, pancreatitis, cancer of the pancreas, stone in the kidney, pericarditis, pleuritis, pneumonia and constipation, even the new infectious pleurodynia, mistaken for coronary thrombosis.

Eventually the differential diagnosis is easy but it may be far from that at any given moment. I am very skeptical about histories of pain and their radiation. As an example, after several internes have finished with a patient with duodenal ulcer this patient will glibly start off and say, "Yes, I have hunger pains associated with an empty stomach and relieved by soda or food." This patient had a very different story when he first went to the

doctor and before he was trained by visits to doctors and hospitals. The description of his pains was not so definitely neat as it afterward became. I have had long discussions with doctors about the precise localization of pain in coronary thrombosis and this is the only safe rule that I know: that in coronary thrombosis, the pain is usually but not necessarily in the chest. It may be in the neck, in the hands, even the right hand, in the abdomen, or there may be no pain at all!

Many of these patients look as if they were sick but that depends upon two factors, the intensity of the pain and the individual. It is sometimes helpful to remember the British classification of typhoid fever, into three classes: the very severe, the moderately severe and the mild. There may be all variations of coronary thrombosis, very mild to very severe. There are some people who are easily upset by pain and others who are not. I have had men in my office who had coronary thrombosis but made light of it and I could not see that it affected their pulse very much, yet after a bit they have become very sick and some have died. One makes the diagnosis of coronary thrombosis in the first instance by thinking of it and in the second instance by eliminating as well as possible the other conditions that need to be considered. If your experience is like mine, you have seen and made mistakes and will continue to make mistakes. But when the case is over, especially if the patient has died, the difficult diagnosis can be made with an emphasis and a conviction that is truly didactic.

One needs to be careful about talking of the prognosis while the patient lives, whether or no the official diagnosis is coronary thrombosis. We may say emphatically that there is no coronary thrombosis, we do not even think that there is any angina. The X-ray shows a small heart, there is a low blood pressure, the electrocardiogram is repeatedly negative. Despite all that, the individual may have rather suddenly an attack which is unquestionably coronary thrombosis and which will result in the proof of the condition at autopsy. Again, the precise converse of this is true. Even with the diagnosis of coronary thrombosis and with the help of X-ray and electrocardiograms, one cannot always be sure as to the extent of cardiac damage.

I do not know to this day whether one patient of mine has had coronary thrombosis or not. He has had various attacks with pain sometimes in his chest, sometimes in his upper abdomen. Half the doctors who have been called in a hurry have said it was angina and coronary thrombosis and the other half called it indigestion. He has a small heart and a low blood pressure. Some of his electrocardiograms could be interpreted as positive and some as negative. One or two of his electrocardiograms, when he was feeling particularly well, have been decidedly poor and any cardiologist would certainly say, "Grave myocardial damage." These abnormalities have all cleared up and he is still living, or was very recently. The awful part of this is that he never has one of the attacks except when he gets indigestion, constipation, some cause of worry and extra excitement and usually something to drink. When he behaves himself from the hygienic point of view, he does very well and is thoroughly convinced that he suffers from nothing more than constipation. I am not ready to make a diagnosis and I refuse to make any prognosis whatsoever. A substantial percentage of such cases eventually turn out to have a dual pathology, for example, gall stones and mild coronary thrombosis. Some of them by successful operation get such remarkably good results that the diagnosis of coronary thrombosis as a real factor in the patient's life must be given up. Too often the eventual outcome, delayed for years of well being, substantiates the accuracy of the diagnosis of coronary thrombosis.

The attacks of coronary thrombosis may be of varying severity, depending upon the situation and extent of the pathologic process. As our knowledge accumulates we are beginning to try to estimate during life not only the extent but the situation of the coronary thrombus. This is possible in certain favorable cases where all the factors seem to show significant findings which all agree. The introduction of a fourth lead and other leads indicate that there are reservoirs of information that we would like to tap but which the usual three electrocardiographic leads do not reach. A patient may have a very stormy time with his coronary thrombosis and seemingly recover and be in excellent health for many years. On the contrary, a seemingly mild attack of coronary thrombosis may be followed by devastating myocardial signs and symptoms. The classical story of coronary thrombosis is persistent anginal pain, usually with vomiting. This pain is not relieved by nitroglycerin and frequently requires several quarters of morphia for relief. After twenty-four hours, there is likely to be a little fever, often accompanied by a poly-

nuclear leucocytosis. In about forty-eight hours one may expect to hear a localized pericardial friction rub. Subsequently, the cardiac rhythm is affected, often violently, beginning with extra systoles, perhaps going into fibrillation and often returning to normal rhythm. The pulse rate with the regular rhythm may be quickened or slowed and the blood pressure, perhaps increased at first, is apt to be much lowered. In severe cases, frequently within the first two or three days, evidence of pulmonary infarction is found but outside of a little cough or a very little bloody sputum these infarctions are apt to be symptomless. Without increased respiration, dyspnoea, or cough, they are often overlooked. It must be remembered that anything may happen and that typical cases are extremely rare. The pain may persist irregularly for twelve to ninety-six hours and yet recovery take place. The more persistent the pain the poorer the prognosis. It has been my experince that young people often have very devastating attacks of coronary thrombosis from which they recover as far as any symptoms are concerned, but that a serious attack of coronary thrombosis in the elderly, around seventy, is a much more momentous affair. I recall well a doctor of sixty odd who had some premonitions of pain on Thanksgiving morning but paid no heed to this and ate a large Thanksgiving dinner. In consequence of that or something else he had a terrific substernal pain which lasted for over forty-eight hours. He had fever and leucocytosis, a pericardial friction rub, large infarctions in his lungs and violent disturbances of his cardiac rhythm. He was in bed for a month and did very little for three months but now, approaching 70, he is back doing full work as general practitioner, climbing stairs, doing obstetrics and everything. His coronary thrombosis occurred in December, 1931. His electrocardiograms have consistently shown myocardial damage and the typical picture of coronary disease. He has not taken particularly good care of himself but has taken digitalis daily. He has now the same gallop rhythm that he had shortly after his attack of coronary thrombosis and the heart's sounds have a sinister thrill.

In only a few cases is the attack of coronary thrombosis more than a severe pain in the chest and a disability of a day or so at the most. The attack of coronary thrombosis which represents what is to the patient necessary illness is relatively rare. Not infrequently one finds that the patient has actually forgotten about his attack of pain although it was so severe that he got some doctor out of bed to give him morphia, but the next day he was well. At a distance it is difficult to say whether it was gall stones, coronary thrombosis or what. In such a case, the electrocardiogram is not infrequently of decisive value. The subsequent devastation of the myocardium does not seem to be dependent upon the storminess of the symptoms but rather upon other factors in the pathology. While the coronary arteries are supposed to be terminal vessels the recent work showing that the myocardium can receive nourishment from the Thebesian vessels is important. As the variation in the Thebesian vessels is very great, it is possible that an individual who has a terminal coronary system may have a definite myocardial scar from a coronary occlusion, while another individual will be able to nourish that portion of the myocardium involved in coronary thrombosis through the Thebesian vessels. It may well be that this fact explains the puzzling discrepancies in some types of cases.

There is no treatment for coronary thrombosis except rest. Of course, there can be no absolute rest for the myocardium. Nitroglycerin usually does not work and morphia is required in a very large dosage. Many men use digitalis from the beginning but it has always seemed to me that the indication for digitalis was congestive heart failure. In cases of coronary thrombosis when congestive failure does occur, digitalis does not seem to be helpful and one would hardly expect that it would because the difficulty is in the heart muscle itself. In the congestive failure subsequent to coronary thrombosis, the diuretics, particularly the newer diuretics containing mercury have been the most valuable. In one case I have used a mercurial diuretic, salyrgan, once or twice a week for a period of three years with excellent results. If I am reasonably sure that a person has coronary thrombosis, I make him stay in bed for a month and then take another month getting up and about. This is a hardship and frequently does no good but it has always seemed to me that it was the only rational routine. My cases which have done the best have followed this procedure. In the storm and stress of the first few days, it is of the greatest importance to have a low calory and low roughage diet. The Karell diet has its place at this stage. I have been very much impressed by the value of this general dietary procedure in all forms of

angina pectoris and in the subsequent treatment of coronary thrombosis. We recognize not only the angina that comes after exercise or after emotional disturbance, made classical by John Hunter when he exclaimed that he was at the mercy of every rascal who chose to annoy him, but also the more common angina that comes on after eating. I have had more success in making these people comfortable with a low calory, low roughage diet with scrupulous care of their bowels by non-gas producing laxatives than by all the other therapeutic measures put together. The use of oxygen, preferably by an oxygen tent and most simply by the handy apparatus of Burgess of this city, is warmly recommended by some. While it is rational to minimize the exertion of respiration, I have not been persuaded of its value as a routine procedure.

After the attack is over and the patient is up and about, I try to be as severe as I can about exercise. I tell the patient that he can use his legs and feet to get him from here to there but he ought to give up walking for exercise and golf. A fair proportion of patients pay no heed to this advice so we are able to test the procedure by trial and error. I am still convinced of the wisdom of advising no exercise. I try to get the individual to give up driving a car but that is not always possible, particularly as the disease is so common among doctors who have to drive themselves. I believe in a low calory, low residue diet. I see no peculiar advantage in complete elimination of alcohol and tobacco or coffee. I recall one gentleman over seventy whose first question was whether his wife or his doctors had communicated with me. I told him they had not and he said, "I am consulting you entirely ethically with their complete permission but I do not want you to be prejudiced in any way." He then told me about an attack of coronary thrombosis that he had had some years before. He had had a very stormy time. He was very glad to be alive and to eschew alcohol, tobacco, coffee and the good things of the table which had been very dear to him. After a year or so he began to think that his wife and the doctors were in a conspiracy. Finally, he insisted upon my acting as a referee as to whether what seemed to him the sole remaining pleasures of life should continue to be kept from him. I readily agreed to a compromise which pleased him but did not please his wife. He had no further attacks of coronary thrombosis and died some three years later of cancer of the stomach.

We need to know a good deal more about coronary thrombosis before we can diagnosticate it with certainty and before we can give a reasonably accurate prognosis, particularly about subsequent attacks. We need to follow many cases through before we can say with conviction that such and such a type of attack of coronary thrombosis will be followed by other and fatal attacks. It seems to be true that the majority of people who have an attack of coronary thrombosis seem to get rid of angina as a symptom in that attack. They may suffer subsequently from congestive failure or sudden heart death but do not commonly have the symptoms of angina pectoris again. There are those that do not recover from their original attack of pain for months and even for years. In many such cases there is a large nervous element but it is possible to assume that some nerve endings, sympathetic if you will, get caught in the thrombotic process and are kept irritated. The common result of an attack of coronary thrombosis is that the individual sooner or later has congestive failure or sudden death. Occasionally an individual is found who has had a well authenticated case of coronary thrombosis who recovers and proceeds to lead a normal, vigorous life. My experience with these men is that they try to push the automobile out of the rut once too often and verify the earlier remark about them.

I have tried to bring before you a different picture of a clinical and pathological entity than you ordinarily receive. I have tried to color this picture by case reports and only regret that the mental picture of these cases which comes continually before my eyes cannot be shown to you. The electrocardiographic data in these cases, important although it is, does not seem to me to be vital. The most valuable time to make the diagnosis is in the first day and that is the time that the electrocardiogram is ordinarily negative. I do not think that this instrument of precision is essential to the diagnosis and treatment of coronary thrombosis.

A profession has for its prime object the service it can render to humanity; reward or financial gain should be a subordinate consideration. The practice of medicine is a profession. In choosing this profession an individual assumes an obligation to conduct himself in accord with its ideals.

From the Code of Ethics of the A. M. A.

THE RHODE ISLAND MEDICAL JOURNAL
106 Francis Street, Providence, R. I.

FREDERICK N. BROWN, M.D., *Editor*
309 Olney Street, Providence, R. I.
CREIGHTON W. SKELTON, M.D., *Business Manager*
ALBERT H. MILLER, M.D., *Assistant Editor*
Contributing Editors
WILLIAM P. BUFFUM, M.D. THAD. A. KROLICKI, M.D.
ALEX. M. BURGESS, M.D. EDWARD V. MURPHY, M.D.
W. LOUIS CHAPMAN, M.D. MALFORD W. THEWLIS, M.D.
G. G. DUPRE, M.D. GUY W. WELLS, M.D.
GEORGE L. YOUNG, M.D.

JAMES WILLIAM LEECH, M.D.

The sudden and tragic death of Dr. James W. Leech has shocked the medical fraternity of Rhode Island; difficult as it is to realize, the stern and immutable fact remains.

Dr. Leech was an outstanding figure in the Rhode Island Medical Society of which he had for twenty years been Secretary, in which he was a tower of strength and wise counselor.

Those of us who were in closest contact with him socially and professionally can only too well appreciate our loss. Our sorrow for his family and our personal grief is beyond anything that can be expressed in words.

History may well write "Dr. James W. Leech, gentleman; alert and proficient in his profession, keen and analytical of mind, dignified and gracious in deportment and bearing, honored by all who knew him, mourned and sadly missed not only by his social acquaintances but by all medical Rhode Island."

And now, alas, to this, our genial and beloved friend, we bid a sad farewell.

F. N. B.

APPRECIATION

By the death of Dr. James W. Leech the Rhode Island Medical Society has lost one of its most valuable members. No man in the state was better qualified to serve the Society and no man devoted time and strength more generously. Dr. Leech was preeminent in vision to realize the value of this Society, in ability to effectively support it, and public spirit to make the necessary sacrifice of energy. Let us hope that many others will emulate him in his efforts to raise our medical societies to a constantly higher level of performance.

W. H. B.

PRAYER

AT DR. JAMES W. LEECH'S FUNERAL

October 9, 1936.

We give thanks to Thee, O God, for this, Thy servant, recalling all in him that made others love him so much. We bless Thee for all the goodness and truth, all the understanding and sympathy, all the friendship and kindness that has passed from his life into the lives of others, and has made the world richer for his presence.

Very many people are thinking of him today,—their hearts full of gratitude for what he has done for them and those dear to them; for what he, in his fine Christian manhood, was to them and to all who knew him. From the midst of his busy days, crowded over full of ministry to human need, he has left us. We shall all be grateful to Thee always for his life and work, and for our great privilege in knowing him. May the quality of spirit which was his enter more fully into our own lives and the life of our community. AMEN.

TO THE AMERICAN COLLEGE OF SURGEONS

On October 16, 1846, at the Massachusetts General Hospital in Boston, William Thomas Green Morton first demonstrated what we now know as Surgical Anesthesia. In the words of William Osler: "Before October 16, 1846, surgical anesthesia did not exist—within a few months it became a world-wide procedure; and the full credit for its introduction must be given to William Thomas Green Morton, who, on the date mentioned, demonstrated at the Massachusetts General Hospital, the simplicity and safety of ether anesthesia." That demonstration is the corner stone of the foundation of Modern Surgery. If it had failed of success, it is unlikely that the American College of Surgeons would now exist.

The October Bulletin of the American College of Surgeons states: "The art and science of anesthesia have rapidly advanced since 1844 and 1846 when Crawford Long and Daniel Morton made their historic discoveries." A misstatement humiliating to all of us, slighting the name of a great benefactor and a topic of professional pride. A comparable error has not been recorded since the

London Dictionary of Dates, in 1851, stated that "the discovery was first made by Mr. Thomas Morton of Boston." For ignorance of the most momentous event in the history of surgery, the Dictionary of Dates might be forgiven, but not the official publication of the world's greatest surgical society.

A. H. M.

PROVIDENCE MEDICAL ASSOCIATION

Minutes of the October Meeting

The regular monthly meeting of the Providence Medical Association was called to order by the President, Dr. William S. Streker, on Monday, October 5, 1936, at 8:45 P. M. The minutes of the last meeting were read and approved. Their applications having been approved by the Standing Committee the following were elected to membership:

Antonio Bellin
Frederic Joseph Burns
Morgan Cutts
Thomas Joseph Dolan
Anne Louise Lawton
Edward Armando Ricci
Edward Francis Ruhmann
Jerome J. Ryan
John Charles Sarafian
Ralph Vincent Sullivan

The President read a letter from Dr. Franklin P. Loury of Newton, Mass., inviting all members interested to the meeting of the Academy of Physical Medicine in Boston on October 20, 21 and 22.

The President announced the appointment of Drs. Fred Coughlin and Joseph Hoey to act as an Obituary Committee for the late Dr. Edward Logan.

The first paper of the evening was read by Dr. William P. Buffum and was entitled "The Role of House Dust in Bronchial Asthma." Dr. Buffum reviewed briefly the current knowledge and the results of important investigations. He described the work and methods employed in the children's O. P. D. of the Rhode Island Hospital, and reported the results obtained in the treatment of 64 patients. Of these 64 patients, treated between January, 1932 and September, 1935, 14% were free from symptoms for one year or longer, 47% were much improved, 31% were improved, and 8% were unimproved. The paper was discussed by Drs. Chafee and Fred Riley.

The second paper was by Dr. J. C. Corrigan of Fall River, Mass., and was entitled "The Incidence and Management of Anemia of Pregnancy." The speaker confined his remarks principally to the hypochronic anemia which is very common in pregnancy and occurs in 25 to 50% of patients, There are three important factors, just as in any such anemia occurring in non-pregnant patients: blood loss, deficient diet, and abnormal physiology of the gastro-intestinal tract. Treatment with adequate doses of iron is very effective. Dr. Corrigan referred to results obtained in giving iron as a prophylactic measure during the last trimester of pregnancy which demonstrated that 25% without iron developed anemia in contrast to 5% of those receiving iron. He also discussed the effects on the blood of infants born of anemic and non-anemic mothers. The paper was discussed by Drs. Hale, Langdon, and F. Riley.

The meeting adjourned at 10:15 P. M.

Attendance 92.

Collation was served.

Respectfully submitted,

HERMAN A. LAWSON, *Secretary*

PAWTUCKET MEDICAL ASSOCIATION

Minutes of the October Meeting

A regular meeting of the Pawtucket Medical Association was held at the Memorial Hospital on October 15, 1936. The President, Dr. W. J. Dufresne, presided. Twenty-two members and five guests attended. Dr. George W. Waterman presented an interesting paper on "Pituitrin—Its Uses and Abuses." A committe was appointed to draw up resolutions on the illness of Dr. Elliott A. Shaw. A committee was appointed to confer with the State Society in regard to some mutual agreement for collection of State Society dues in compliance with the new by-law of the Pawtucket Medical Association. Meeting adjourned at 10:15 P. M. Collation was served.

Respectfully submitted,

THAD. A. KROLICKI, M.D.,
Secretary.

Personal Notes

All other events are overshadowed by the unwelcome news of the death of Dr. James W. Leech, Vice President and long time Secretary of the Rhode Island Medical Society. At the Jane Brown Hospital, on the morning of October 6, while on his customary round of duty, he was suddenly stricken with a fatal angina. The only warning had been a slight similar attack six months before.

The funeral service was held at noon, Friday, October 9, at the Central Congregational Church in Providence, of which Dr. Leech was a Deacon. It was attended by a multitude of physicians, patients and friends. The service was conducted by Reverend Arthur H. Bradford, Minister of the Church, assisted by Miss Elizabeth Bugbee at the great organ. The ushers were Dr. F. M. Adams, Dr. E. S. Brackett, Dr. H. G. Calder, Dr. N. H. Gifford, Dr. B. H. Buxton and Mr. Edward A. Stockwell. The bearers were Dr. J. E. Donley, Dr. A. H. Ruggles, Dr. D. L. Richardson, Dr. W. O. Rice, Dr. H. G. Partridge, Dr. H. C. Messinger, Dr. Halsey DeWolf and Kirk Smith, Esq. The Rhode Island Medical Society was officially represented by Drs. Rocheleau, Hawkins, A. T. Jones, Wells, and W. S. Streker, and the Providence Medical Association by past and present officers; Drs. Kingman, Gormly, Donley, Buffum, and Chase.

The East Providence Physicians' Association was organized in January, 1936. Five meetings have been held. At the July meeting a Code of By-Laws was discussed and adopted. Objectives of the Association are a better understanding among the members and a much broader and more sympathetic understanding between local physicians and the various agencies with whom they deal.

Much attention has been given to local welfare problems, among them the following:

1. Proper examination of children before entering school.

2. General revision of the work of school physicians, in order to secure more satisfactory health supervision and avoid undue sacrifice of the physicians' time.

3. Arrangement of clinics for vaccination of children about to enter school.

4. Co-operation with State agencies in the conduction of those clinics in which said agencies are interested.

The officers are: Dr. James Moore, President; Dr. Theodore C. Hascall, Vice-President; Dr. Harrison F. Hyer, Secretary-Treasurer.

Sunday, October 4. At the dedication of the first buildings to be completed at the State Hospital for Mental Diseases at Howard, the principal address was delivered by his Excellency, Governor Theodore Francis Green. The introductory address was made by Rhode Island Medical Society President, Dr. John E. Donley, State Director of Public Welfare, in his usual pleasing and effective style. The exercises were held on the grounds in front of the new women employees dormitory, and opposite the new auditorium. An efficient loud speaker system enabled all the audience to readily listen to the speakers.

October 15. A regular meeting of the Friday Night Medical Club of Providence initiated the forty-ninth season of the activity of the club. Dr. J. Murray Beardsley, guest speaker, gave an interesting address on "Surgery of the Chest." Among many phases of his subject, he laid special stress on the value of artificial pneumothorax and partial thoracoplasty in treatment of pulmonary tuberculosis. The subject was thoroughly discussed.

October 19. At the meeting of the Thirty-four Medical Club, Dr. Francis H. Chaffee read an interesting paper on "Snake Venom." The subject was well discussed by members of the club.

October 20. The first lecture in the series arranged for the General Staff meetings of the Homeopathic Hospital of Rhode Island was given by Dr. Reginald Smithwick of the Massachusetts General Hospital. His subject was "Sympathectomy in Vascular Disease with Special Reference to Essential Hypertension."

October 28. At a regular meeting of the Jacobi Club, Dr. I. Gerber spoke on "X-Ray Treatment of Infections and other Inflammatory Processes."

October 29. The Rhode Island Medico-Legal Society held a regular Quarterly Meeting at the Medical Library. The speaker was Joseph H. Hagan, Chief of Division of Probation and Parole, State Department of Public Welfare; his subject, "Understanding the Delinquent."

Wednesday, October 14. An overcast sky did not dampen appreciably the enthusiasm at the Third Annual Golf Tournament of the Providence Medical Association and the Rhode Island Bar Association, which was played at the Wannamoisett Country Club. While the lawyers took the individual honors, the doctors won the tourney by 71 strokes and hold for another year the cup, put up for competition by James C. Collins, Esq. and Dr. Charles F. Gormly in 1934. At the dinner which followed the play was reviewed with the same spirit of generous rivalry which had characterized the tournament. Attendance, 61.

The score:

DOCTORS

Frank Honan	45	43	88
E. F. Burke	49	40	89
R. F. McCoart	44	45	89
B. Feinberg	43	47	90
C. O. Cooke	47	44	91
C. D. Sawyer	47	45	92
F. A. Webster	47	46	93
E. S. Brackett	49	46	95
R. R. Hunt	51	45	96
E. I. Seltzer	49	48	97
B. H. Buxton	45	52	97
David Freedman	51	46	97
E. G. Melvin	47	51	98
R. H. Whitmarsh	51	48	99
A. Archetti	47	53	100
J. M. Gibson	48	53	101
M. J. Nestor	53	48	101
V. J. Ryan	53	48	101
S. G. Lenzner	47	55	102
F. A. Coughlin	49	55	104
R. F. Hacking	55	54	109
W. C. Gordon	58	51	109
Dr. A. A. Barrows	53	56	109
N. A. Bolotow	58	52	110
M. Goldberger	51	59	110
J. C. O'Connell	57	54	111
W. P. Buffum	54	62	116
A. F. McAlpine	63	56	119
K. K. Gregory	63	59	122

LAWYERS

H. B. Tanner	40	43	83
E. T. Voight	43	41	84
R. M. Greenlaw	43	42	85
M. Addeo	43	44	87
R. C. Green, Jr.	47	42	89
F. A. Otis	44	44	88
J. B. Linehan	47	47	94
H. A. Clason	44	50	94
O. L. Heltzen	44	51	95
R. H. W. Hankins	45	51	96
D. H. Morrissey	44	53	97
W. V. S. Sumpter	50	49	99
L. S. Walling	47	56	103
Nathan M. Wright, Jr.	54	53	107
R. E. Allen	53	55	108
C. E. Wheeler	51	59	110
H. C. Hart	59	53	112
W. H. Strauss	56	57	113
L. A. Worrall	58	61	119
R. G. E. Hicks	62	64	126

Rhode Island Hospital Notes

Dr. Ralph Curtis Farrington, who completed his internship in December, 1935, and also interned for 6 months at the Providence Lying-In Hospital, has opened an office for general practice in Framingham, Mass.

On October 6th, Dr. Nathaniel Beaver, Night Superintendent since July 1st, 1935, accompanied by Mrs. Beaver, left for his former home in Walla Walla, Washington, where he intends to enter private practice. Dr. Beaver's internship terminated May 1st, 1935.

Dr. Jerome J. Ryan, Providence College, 1928, McGill Medical School, 1933, who completed his internship in September, 1935, has been substituting as Night Superintendent and House Physician for the past 4 months. Dr. Ryan has opened an office for practice of Medicine and Surgery at 209 Elmwood Avenue, Providence.

Dr. Richard Bruning completed his internship October 1st, 1936. Dr. and Mrs. Bruning have taken up residence in the Westminster Apartment, Westminster Avenue, Elizabeth, N. J. Dr. Bruning has opened his office in the same building.

Dr. Frederick Alton Webster, of Beverly, Mass., Harvard, 1927, and Tufts Medical School, 1933, became Night Superintendent at the R. I. H. on October 1st, 1936. Dr. Webster was an intern from September, 1933, to October, 1935. For the past year he has held a teaching fellowship at the Boston Dispensary.

Dr. Forrest Martin, intern at the R. I. H. from April, 1934, to May, 1936, after a visit to his home in Missouri, became an intern at the Massachusetts Eye and Ear Infirmary, Boston. Dr. Martin recently paid a short visit to the R. I. H.

Dr. Reginald Farrow, whose internship terminated in 1934, is at the Ruptured and Crippled Hospital in New York City. He recently paid a short visit to the R. I. H.

Dr. Byron L. Sweet, Yale Medical School, 1936, began internship on October 15th, 1936. Dr. Sweet's home is in Tarrytown, N. Y.

Dr. Heinz Lorge, for nine months Resident Cardiologist at the R. I. H., on October 1st entered St. Vincent's Hospital, Worcester, Mass., on a rotating internship.

Married: October 1. Dr. John Egoville to Miss Liboria Marceca, in Brooklyn, N. Y.

Born: October 11. To Dr. and Mrs. Gordon J. McCurdy, a son.

Among the large attendance at the 26th Annual Clinical Congress of the American College of Surgeons, Philadelphia, October 19-23, were Drs. J. L. Belliotti, N. A. Bolotow, E. S. Cameron, P. P. Chase, W. B. Cutts, W. P. Davis, R. DiLeone, F. S. Hale, A. H. Jackvony, L. C. Kingman, W. M. Muncy, J. C. O'Connell, V. J. Oddo, H. C. Pitts, E. M. Porter, T. F. Scanlon.

Drs. J. A. Hayward, A. H. Miller, and M. Saklad attended the co-operating Congress of Anesthetists and received Certificates of Fellowship in the International College of Anesthetists.

RECENT BOOKS

ALLERGY OF THE NOSE AND PARANASAL SINUSES, a Monograph on the Subject of Allergy as Related to Otolaryngology, by French K. Hansel, M.D., M.S., Assistant Professor of Clinical Otolaryngology, Washington University School of Medicine; Fellow of the Association for the Study of Allergy, the Association of Resident and Ex-Resident Physicians of the Mayo Clinic, the American Laryngological, Rhinological and Otological Society, and the American Academy of Ophthalmology and Otolaryngology. With fifty-eight text illustrations and three color plates. The C. V. Mosby Company, St. Louis, 1936. Price $10.00.

This is a very excellent monograph on the subject of allergy as related to the field of otolaryngology. Since the birth of otolaryngology as a specialty some 50 or more years ago, attention has been directed chiefly to the anatomy, clinical aspects and perfection of operative procedures. However, about 5 years ago, Proetz pointed out that the results by surgical and non-surgical methods were far from satisfactory. It was apparent that there was a great need for better understanding of the physiology, biochemistry, pathology, immunology and experimental surgery of the paranasal sinuses, in order to establish better methods of diagnosis and treatment. In this book the author very carefully discusses the fundamental principles of physiology, biochemistry, bacteriology of the secretions, the cellular reactions of the tissues in allergy and in immunity, and the histopathology of allergy as they relate to the nose and paranasal sinuses.

He mentions the need of a thorough understanding of allergy for every otolaryngologist, in order that he may diagnose and successfully treat inflammatory diseases of the nose and sinuses. Many of these are allergy, and others are a combination of allergy and infection. The high incidence of allergic manifestations in the nose also suggests a basis for other accompanying symptoms.

Allergy is discussed in detail as to its origin and development. The fundamental clinical characteristics with variations are considered, as well as methods of testing with the real value of each, in the experience of various groups of men.

Some space is given to bronchial asthma in its many phases. Methods of treatment are enumerated, especially the results of radical surgery of the sinuses and those treated more conservatively. A chapter is given on the part bronchoscopy plays in asthma.

A summary is made of the great importance of the part played by allergy among the diseases of the upper and lower respiratory tracts in children in its relation to both otolaryngological and pediatric conditions, in order to establish a diagnosis.

A chapter is devoted to allergy and immunity in opthalmology. The treatment of retro-bulbar neuritis by the intravenous injection of triple typhoid vaccine is discussed by Benedict. He believes that any operation on the sinuses is unwarranted unless evidence of suppurative disease is established.

Finally the treatment of allergy and hay fever is given in great detail, condemning certain methods and enlarging on the correct ones. A number of illustrative cases of clinical allergy are reviewed to point out some of the principles of diagnosis and treatment.

There are many quotations from the literature and an extensive bibliography.

L. C. H.

SYNOPSIS OF DISEASES OF THE HEART AND ARTERIES. By George R. Hermann, M.D., Ph.D., Professor of Clinical Medicine, University of Texas. Pp. 344 with 88 Text Illustrations and 3 Color Plates. Cloth, $4.00. The C. V. Mosby Company, St. Louis, 1936.

In the preface the author, an outstanding teacher and investigator, states that "This synopsis is an attempt to provide an acceptable indexed epitome of the principles and modern conceptions of cardiologic practice. It is in no sense of the word a book for the specialist."

In general, the author has succeeded in his purpose admirably. The book, 328 pages in length, contains almost all the practical information found in most larger texts. Clear, up-to-date information concerning history taking, physical examination and the value of various laboratory procedures is afforded. Ample discussion of differential diagnosis, prognosis and treatment should make the book of value to the practitioner as a ready source of useful information.

The book is compact and is adequately though not profusely illustrated. The presentation of the subject matter is clear and most of the recent additions to diagnosis and therapy in heart disease are described.

One minor criticism might be that no mention whatsoever is made of the heart failure associated with beri-beri and arteriovenous aneurysm, both conditions, though rare, offering the possibility of complete cure. However, the merits of the book are so numerous that it can be recommended without reservation.

F. B. C.